

FcRn-BASED THERAPEUTICS FOR THE TREATMENT OF AUTO-IMMUNE
DISORDERS

Abstract of the Disclosure

Disclosed is a transgenic knockout mouse whose genome
5 comprises a homozygous disruption in its endogenous FcRn
gene, wherein said homozygous disruption prevents the
expression of a functional FcRn protein, resulting in a
transgenic knockout mouse in which exogenously administered
IgG1 exhibits a substantially shorter half-life, as compared
10 to the half-life of exogenously administered IgG1 in a wild-
type mouse. Also disclosed is a transgenic knockout mouse
whose genome comprises a homozygous disruption in its
endogenous FcRn gene, wherein said homozygous disruption
prevents the expression of a functional FcRn protein,
15 resulting in a transgenic knockout mouse which is unable to
absorb maternal IgG in the prenatal or neonatal stage of
development. Methods of using the transgenic knockout
mouse, and cells derived therefrom, are also disclosed.